Factors regulating ventricular rates during atrial flutter and fibrillation in pre-excitation (Wolff-Parkinson-White) syndrome

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Premature atrial stimulation and His bundle recordings were performed in 6 patients with pre-excitation (WPW) syndrome. The ventricular rates during bursts of atrial flutter and atrial fibrillation triggered by the earliest impulses depended on the functional properties of the accessory pathway. In 3 patients in whom the effective refractory period of the accessory pathway was longer than that of the normal AV pathway the ventricular rates did not exceed 210/min; the majority of the supraventricular impulses reached the ventricles through the normal AV pathway. In 3 other patients in whom the effective refractory period of the accessory pathway was very short (less than 220 msec) the ventricular rates were as high as 310/min; the majority of the impulses reached the ventricles through the accessory pathway. Concealed AV conduction occurred in both pathways producing RR pauses well in excess of the duration of the corresponding effective refractory periods. These tachyarrhythmias were due to stimulation during the atrial vulnerable period, not to a reciprocating mechanism involving two separate anatomical AV communications.

Intracardiac recordings were necessary to evaluate the AV conduction patterns as well as the nature of the wide QRS complexes seen during the tachyarrhythmias.

Factors regulating the ventricular rates during atrial flutter and fibrillation in patients with pre-excitation (WPW) syndrome are not well known. Previous studies from our department suggested that the AV conduction patterns occurring during these tachyarrhythmias depended on the duration of the effective refractory period of the accessory pathway and on its relation to that of the normal pathway (Castellanos *et al.*, 1973).

The present communication corroborates these assumptions.

Subjects and methods

The technique for obtaining His bundle recordings used in our department has been discussed previously (Castellanos and Castillo, 1972; Castellanos *et al.*, 1973). After consent was obtained from the patient and discontinuing all drugs for at least three days, premature atrial stimulation and His bundle recordings were performed during a basic paced atrial drive in 6 patients with the type of pre-excitation (WPW) syndrome in which the forward H deflection was inscribed within the QRS complex. During the course of the study bursts of atrial flutter and

fibrillation were triggered by some of the most premature atrial stimuli. The analysis of these arrhythmias constitutes the basis for this report.

The effective refractory period of the accessory AV pathway was given by the longest A_1–A_2 interval at which A_2 failed to produce a QRS complex with a delta wave (Castellanos *et al.*, 1973). The effective refractory period of the normal AV pathway was given by the longest A_1–A_2 at which A_2 failed to produce a 'fusion' or 'combination' QRS complex (Castellanos *et al.*, 1973).

Results

There were 3 patients in whom the effective refractory period of the accessory pathway was longer than that of the AV node (Table 1). At long coupling intervals AV conduction occurred through the accessory pathway, but once a given interval was reached propagation to the ventricles occurred exclusively via the normal AV pathway. Very premature stimuli triggered runs of atrial flutter and fibrillation during which nearly all impulses reached the ventricles exclusively through the normal AV pathway. Occasional fusion beats were noted. Exclusive accessory pathway conduction was

TABLE I *Data on 3 patients in whom effective refractory period of accessory pathway was longer than that of normal pathway**

| Case No. | WPW type | Effective refractory period | | Driving cycle length of high rt. atrium |
		Accessory pathway	Normal pathway	
1†	B	350	265	640
2	A	410	310	680
3	A	360	315	600

* All values in milliseconds.
† For further information see Case 1 of the article by Castellanos *et al.* (1973).

TABLE 2 *Data on 3 patients in whom effective refractory period of accessory pathway was extremely short, presumably shorter than that of normal pathway**

| Case No. | WPW type | Effective refractory period | | Driving cycle length of high rt. atrium |
		Accessory pathway†	Normal pathway	
4‡	B	220	—	600
5§	A	190	—	750
6**	A	210	—	850

* All values in milliseconds.
† Effective refractory period shorter than these values.
‡ For further information see Case 2 of the article by Befeler *et al.* (1973).
§ For further information see Case 2 of the article by Castellanos *et al.* (1973).
** For further information see Case 4 of the article by Castellanos *et al.* (1973); and Case 3 of the article by Castillo *et al.* (1973).

rare in these cases (Fig. 1). The ventricular rates did not exceed 210/min. These arrhythmias were of short duration and disappeared spontaneously.

In 3 other patients (Table 2) the effective refractory period of the accessory pathway was very short (less than 220 msec). Its exact duration could not be determined in Cases 4 and 6 because of the occurrence of persistent tachyarrhythmias, and in Case 5 because the atria became effectively refractory while conduction was still occurring through the accessory pathway. Neither could the effective refractory period of the normal AV pathway be measured with

accuracy since the coupling interval at which a fusion QRS complex failed to occur could not be determined. This was due to the fact that at short coupling intervals the supraventricular impulses reached the ventricles while the latter were incompletely recovered, therefore causing some
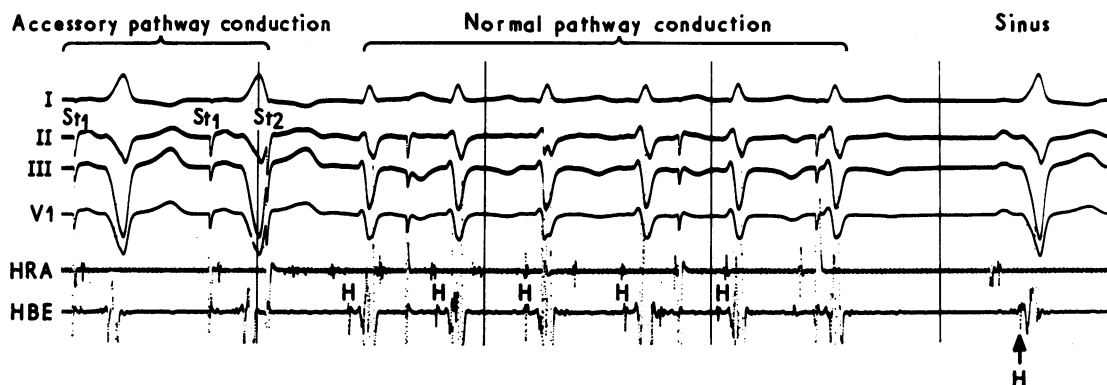

FIG. I *(Case 1) Short run of atrial flutter (?) in a patient with WPW type B in which the effective refractory period of the accessory AV pathway exceeded that of the normal AV pathway. Driven impulses (St1) were conducted exclusively or predominantly through the accessory pathway (first two QRS complexes). The forward H deflection was lost within the ventricular electrogram of the HBE lead. A premature stimulus (St2) delivered at a coupling interval of 250 msec triggered a short run of atrial fibrillation or atrial flutter which subsided spontaneously. During the tachyarrhythmia AV conduction occurred exclusively through the normal pathway as determined by the presence of an H deflection in front of the QRS complexes without a delta wave and a left anterior hemiblock morphology. The HV interval was of normal duration. The last beat was a fusion complex produced by a P wave of sinus origin. Note that the forward H deflection was inscribed after the onset of depolarization. HRA = bipolar electrogram from the high right atrium. HBE = His bundle electrogram. In this and all figures (except Fig. 2) paper speed and time markers are 100 mm/sec and 1 sec, respectively.*

distortion of the ventricular complexes. It was not possible to determine the coupling interval at which a fusion beat (resulting from AV conduction through both pathways) ended and when aberration due to incomplete ventricular recovery started. However it is unusual for the effective refractory period of the normal pathway to be less than 220 msec (Wit *et al.*, 1970).

In these 3 patients (Cases 4, 5, and 6) the ventricular rates during the arrhythmias were as high as 310/min. Nearly all supraventricular impulses reached the ventricles exclusively, or predominantly, through the accessory pathway. The resulting QRS complexes either had a 'pure' WPW contour, or a WPW morphology distorted by incomplete ventricular recovery. Fusion beats were also noted. Occasional impulses propagated exclusively through the normal AV pathway.

All 3 patients showed periods in which the RR intervals were as long as 600 msec. During these pauses AV conduction did not occur through either pathway, presumably because of concealed conduction in both structures.

The atrial arrhythmias in Cases 4, 5, and 6 did not disappear spontaneously. They required transthoracic electrical conversion for restoration of sinus rhythm.

Discussion

Mechanisms of atrial flutter and fibrillation in pre-excitation (WPW) syndrome

Tachyarrhythmias occurring in patients with WPW syndrome can be due to several mechanisms. For
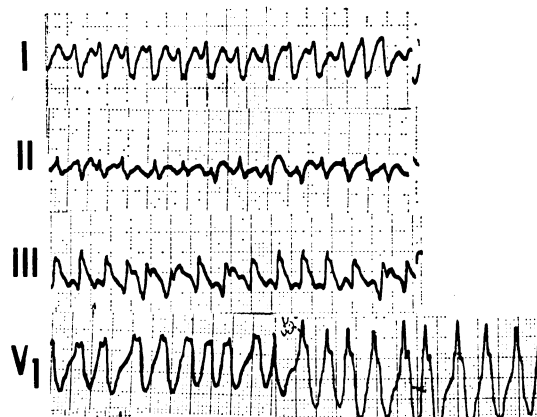

FIG. 2 *(Case 5). Persistent atrial fibrillation in a patient with WPW type A in which the effective refractory period of the accessory pathway was shorter than 190 msec. The supraventricular impulses reached the ventricles exclusively, or predominantly, through the accessory pathway. The ventricular rates were as high as 310 × min.*

instance, in 1964 Scherf and Cohen suggested that a reciprocating rhythm involving two separate anatomical pathways (Kent bundle and normal AV junction) could not explain the occurrence of atrial fibrillation.

The newer techniques of atrial pacing and His bundle recordings have shown that premature stimuli delivered early in the cycle (soon after the

FIG. 3 *(Case 4). Persistent atrial fibrillation in a patient with WPW type B in which the effective refactory period of the accessory pathway was shorter than 220 msec.*

All supraventricular impulses (except one) reached the ventricles exclusively through the accessory pathway. The third QRS complex from the end of the strip was conducted to the ventricles through the normal pathway with some degree of right bundle-branch block. The relatively long RR interval (630 msec) preceding this ventricular complex occurred because of the concealed penetration (of supraventricular impulses) into both pathways. Though some of the sharper deflections recorded by the His bundle electrogram lead might have been H deflections the majority were atrial in origin. The fastest ventricular rate recorded in this patient was 270 × min.

end of the atrial effective refractory period) could initiate runs of reciprocating tachycardia as well as of flutter or fibrillation (Castillo and Castellanos, 1970). The latter were induced by the electrical stimuli falling in the vulnerable period of the atria. Whereas reciprocating tachycardia was terminated by vagal manoeuvres and properly timed electrical stimuli (Castillo and Castellanos, 1970) atrial fibrillation disappeared either spontaneously, or required electrical conversion. In earlier studies it was believed that persistence of the latter arrhythmia was related to the presence of coexisting atrial disease (Castillo and Castellanos, 1970). Nevertheless the events in Cases 4 to 6 suggested that they might persist in patients without clinically or haemodynamically detectable atrial diseases provided that the effective refractory period of the accessory pathway is very short (Table 2 and Fig. 2–6). This is in contrast to what occurs in the cases in which the effective refractory period of the accessory pathway is relatively long (Table 1 and Fig. 1). The reasons for this behaviour of the fibrillating atria are not clear.

AV conduction patterns and ventricular rates during atrial flutter and fibrillation in pre-excitation (WPW) syndrome

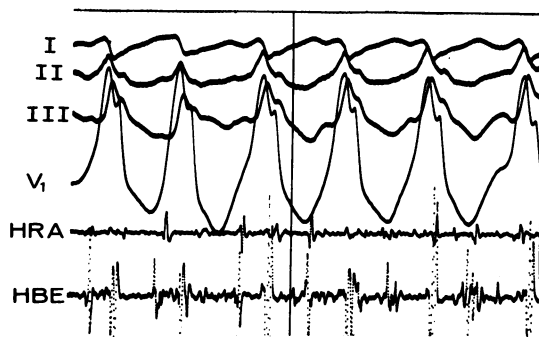Several authors have observed that the ventricular rates in atrial flutter and fibrillation can vary from very fast (pseudoventricular tachycardia [Herrmann et al., 1957]) to relatively slow (between 90 and 120 msec) (Katz and Pick, 1956; Laham, 1969).

Conduction through both pathways has been known to alternate but the reasons why the rates could vary so much have been a matter of debate and speculation. The present communication and a previous study (Castellanos et al., 1973) suggested that ventricular rate depended on the duration of the effective refractory period of the accessory pathway. When it was longer than that of the normal AV pathway (and AV nodal block was not present) the majority of the impulses were transmitted across the normal AV pathway through the
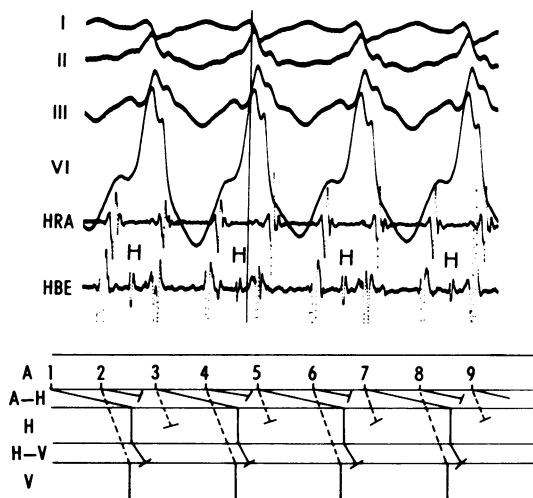

FIG. 5 (*Case 6*). *Atrial flutter with 2:1 AV conduction (to the ventricles) exclusively through the accessory pathway. Note that all H deflections are inscribed after the onset of ventricular depolarization. The atrial rate was 300 × min. Numbers at the A level indicate consecutive low right atrial impulses.*

Broken oblique lines extending from A to V represent conduction through the accessory pathway. Solid lines at the AH level indicate propagation through the AV node. The tracings show 2:1 AV conduction through the accessory pathway since A_2, A_4, A_6, and A_8 were conducted, while A_1 (not shown) A_3, A_5, A_7, and A_9 failed to do so, either because the accessory pathway was effectively refractory or because the impulses 'decremented' through it. In addition there was 2:1 AV block at the AV node. A_1 (not shown), A_3, A_5, and A_7 activate His bundle (thereafter being unable to depolarize the ventricles due to the ventricular refractoriness created by the impulses traversing the accessory pathway). However A_2, A_4, A_6, and A_8 were not followed by H deflections indicating that they were blocked within the AV node.

FIG. 4 (*Case 6*). *Atrial fibrillation in a patient with WPW type A in which the effective refractory period of the accessory pathway was shorter than 220 msec. Information regarding this patient (specifically the QRS morphology resulting from exclusive accessory pathway conduction) can be found in previous articles (Castellanos et al., 1973; Castillo et al., 1973).*

All supraventricular impulses reached the ventricles exclusively through the accessory pathway. The distortion created by the fibrillating atria in the His bundle electrogram lead made (as in Fig. 3) the identification of H deflection impossible.

FIG. 6 (*Case 6*). *Atrial flutter with varying AV conduction through both pathways. The first four QRS complexes resulted from exclusive accessory pathway conduction. During this part of the tracing there was 2:1 and 3:1 accessory pathway conduction alternating with 5:1 and 4:1 conduction between atria and His bundle (through the AV node). A_1 and A_6 were unable to activate the ventricles because the latter had already been depolarized by the impulses traversing the accessory pathway. The last three QRS complexes resulted from exclusive normal pathway conduction with block in the right bundle-branch. They were preceded by H deflections with normal HV intervals. During this part of the tracing there was 2:1 AV conduction through the normal pathway. None of the impulses propagated through the accessory pathway. The 2:1 and 3:1 pattern that (at the beginning of the tracing) had been occurring through the accessory pathway could no longer occur because A_{10}, A_{12}, and A_{14} reached the ventricles slightly ahead of those impulses (A_{12} and A_{14}) which otherwise would have been totally responsible for the production of the QRS complexes.*

AV node. Concealed conduction through the AV node produced enough R to R prolongation of consecutive narrow QRS complexes to allow conduction through the accessory pathway. On the other hand, when the effective refractory period of the accessory pathway was very short the atrial impulses reached the ventricles through the accessory pathway. Concealed conduction through this pathway resulted in long RR cycles terminated either by anomalous, or normal, QRS complexes (depending on the presence of coexisting concealed conduction through the AV node).

A thorough evaluation of concealed conduction required the recording of His bundle electrograms and knowledge of the functional properties of both pathways. That the effective refractory period of the accessory pathway was relatively long in some cases, while being extremely short in others, has been attributed to differences in the physiological properties of the accessory pathway. It appears as if in the former instances this structure behaved

(functionally) as Purkinje tissue, whereas in the latter it resembled ordinary atrial muscle. However, this assumption requires corroboration by further electrophysiological and anatomical studies.

Wide QRS complexes during atrial flutter and fibrillation in pre-excitation (WPW) syndrome

Intracardiac recordings were necessary to determine the nature of wide QRS complexes seen during the tachyarrhythmias. Whereas some beats were pure, or fusion, WPW complexes, others resulted from exclusive accessory pathway conduction with distortion created by propagation during the period of incomplete ventricular recovery. Reaching the correct diagnosis required knowledge of the QRS morphology produced by exclusive accessory pathway conduction (in which the forward H deflection was inscribed after the onset of ventricular depolarization) and of that resulting from exclusive normal AV pathway conduction with, or without, bundle-branch block in which the forward

H deflection was inscribed at its normal time in front of the ventricular complexes.

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